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TABES IN THE NEGRO.*

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About the negro much has been set down in the annals of anthropology, ethnology, and sociology, and a glance at medical literature as it appears in summary form from year to year would suggest that almost the last word has been written about tabes; but of tabes in the negro I have been able to obtain only the fewest references recorded, in the main, by American writers. Von Leyden[1] quotes Buri as sponsor for the statement, "*bei den so häufig syphilitischen Negern soll die Tabes sehr selten vorkommen*" (tabes occurs very rarely in the large number of syphilitic negroes). In another foreign contribution to the literature of this subject, entitled "*La Ataxia Locomotriz* (*en el negro*),"[2] is recorded the fact of tabes occurring eight times in full-blooded negroes and three times in mulattoes.

C. W. Burr[3] in a report on "The Frequency of Locomotor Ataxia in Negroes," read before the Philadelphia Neurological Society, 1892, prefaces the comments on his own case with the written opinions of many Southern practitioners, all of whom were unanimous in declaring tabes in the negro of rare occurrence. Professor Osler is also quoted, in answer to Dr. Burr's query, as stating "of twenty cases under observation since the opening of Johns Hopkins Hospital, one was a full-blooded negro and one a mulatto." Dr. Potts, physician to the Nervous Dispensary of the University of Pennsylvania, told Dr. Burr that he had not seen a case in eight years. During fourteen years, says Dr. Burr, not a case applied at the Infirmary for Nervous Diseases in Philadelphia. He adds: "For my own part, I have never seen a case in the pure black, and but one in a mulatto." McConnell[4] presented a report of five cases before the Philadelphia Neurological Society in 1899, in which he stated that he had seen "tabes in many whites, a few cases among the mixed races, and but five instances in *pure* negroes." Four of these cases were in males and one in a female. Dr. J. H. Lloyd,[5] in 1896, reported one case in a negress. In a tabulation of 194 cases of tabes, with a view to demonstrating the distribution among the different nationalities, F. Savary Pearce[6] gives the following:

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* Read at the meeting of the Chicago Neurological Society, October, 1903.

American and English	119
French	2
German	21
Hebrew	6
Irish	25
Colored (not pure)	2

The next reference in American literature, and one of more recent date, is that of A. P. Francine,[7] who discusses briefly "Two Cases of Tabes Dorsalis in Negroes—Husband and Wife." In this article S. Weir Mitchell is credited with saying that he has seen four cases of tabes, two of which had occurred in mulattoes in Florida.

Aside from these allusions in the literature to the subject-matter of this paper, it has become, I believe, almost a habit with clinicians to regard the colored people as exempt from this disease. Neurologists as well, who from year to year attract an ever-increasing, vast clinical material, confess to having seen little or no tabes in the negro. My chiefs in the Department of Nervous Diseases at the Northwestern University Medical School, Professors Hugh T. Patrick and Archibald Church, record having met with three cases each in their entire experience.

It has been my good fortune during the past year to see and study four undoubted cases of tabes in colored individuals, three of them being in females and one in a male. Although adjudged in the light of sheer coincidence, this ratio of three to one in favor of the female is unusual, since statistical plurality is quite the other way. The clinical picture in these cases in no way deflects from the symptom-complex we are accustomed to see in the whites except, perhaps, in the fact that they are all of the "amaurotic type" of the disease. Satisfied on the score that there is nothing of a pathological nature to reveal, I am, nevertheless, mindful of other points of interest which I believe should command the attention of medical men, and neurologists in particular, and for the following reasons:

1. The relative vital movement of different races in a mixed population is a matter of interest to the physician as well as the ethnologist.
2. The white and colored races living side by side in this country and in some of the Southern States in nearly equal numbers afford an opportunity for study, though the problem is rendered complicated yet interesting by their partial admixture.
3. The compilations and statistics in the past on " race and disease " have slighted certain diseases of which we are in duty bound to know more than we do, in order to avert inadequate and incomprehensive conclusions.
4. Tabes in the pure and hybrid negro should afford fresh incentive for a study and investigation of the, in large part accepted, in part conflicting, and in part rejected, evidence of a luetic etiology.
5. The influence of race amalgamation on production and inhibition of disease (with special reference to civilization and syphilization as a factor in the production of tabes) is important in all lands.

To quote from that brilliant and exhaustive treatise, " The Surgical Peculiarities of the Negro," written by Dr. Rudolph Matas,[8] of New Orleans, and delivered at the 1896 meeting of the American Surgical Association, " the influence of crossing and a comparative study of the negro is a prerequisite to the understanding of medical shades of difference."

These questions can only be approached by a better understanding of the vicissitudes through which the negro has passed since the days of his importation from Africa, but an historical commentary treating of the native African negro would far exceed the intended scope of this paper. Concerning negroid ancestry in Africa, I might say that valuable accounts of the endemic diseases afflicting the negroes who live in semisavage or tribal state in Africa appeared in 1895 in communications to the German Government by Dr. A. Plehn.[9] He dwells at length upon all the diseases to which the negro is prone, briefly classifying the nervous diseases as follows:

" Beriberi prevails in Kamerun, but perhaps to a greater degree in Sierra Leone and Congo. I have seen a single case of hysteria (*einen ausgesprochenen Fall*), with all its signs and symptoms, in the wife of King Bell. Epilepsy occurs; I recall four cases (*ausgeprägte Fälle*) in Kamerun." This exhausts his list.

When the negro was first imported he brought with him diseases peculiar to himself (*sui generis*) and foreign to the inhabitants of our colonies; yaws—the sleeping sickness—and elephantiasis belonged prominently to the earlier era in this country. A study of these and other diseases was immediately begun by Southern practitioners, and the numerous authentic works extant as classics bear witness to the untiring effort and careful observation made by the Southern profession. The names of Nott (physician and anthropologist), Gliddon, Cartwright, Grier, Corson, and Tipton are inseparably linked with the literature exploited in the Southern medical journals as far back as 1850. Trismus nascentium—marasmus—referred to by Grier as " literally a wasting away—a tabes of young life—provincially styled ' the drooping disease of negro children,' " invermination—cachexia Africana—(dirt-eating disease of the negro) are familiar topics in the pioneer literature of the South. Grier,[10] in 1852, said: " Whether the races be derived from one or more original species, they are now characterized by such peculiarities of constitution and temperament as to make the distinctive maladies of the negro race a subject of much practical importance to us and worthy of a more painstaking investigation than it has ever yet received from the hands of the medical profession of the South."

According to the consensus of medical and statistical opinion, release from bondage has been the turning-point in the negro's physical welfare. Such medical writers as Tipton, Furniss, and principally Corson and Matas, pronounce this as the beginning of his downfall, his physical deterioration and moral degradation. Since the war and emancipation the avenues of independent occupation have been flung wide open to irresponsible beings, unprepared for their unequal lot. The introduction of civilized vices into uncivilized communities began anew, and the negro was quick to accept and devour the vices and excesses of material civilization. Out of the general morbidity and depravity of this new community of interests sprang a mortality record which was immediately higher than that of the whites in ante-emancipatory times and has been getting increasingly greater in later years, until now the nation feels a keen concern in the gravity of the " vital statistics of the negro."

Diseases incident to the state of slavery are disappearing, and tuberculosis, syphilis, and cancer are deep-rooted in the colored people. Old immunities are lost. Admixture with white blood has weakened the race. In the presence of new blood has come the tendency to acquire new diseases; the amalgamation is responsible for much that is radically new in the pathological field.

By a legitimate intermingling of tribal strains of blood originally distinct in Africa and an illegitimate, universal miscegenation with the whites, the typical American negro of to-day has incorporated in his formation certain generic characteristics of his negroid ancestry plus the deteriorating phenomena of his hybridity.

CASE HISTORIES. Whereas, the cases I am about to report do not present in number or variety the symptoms we are accustomed to associate with this essentially chronic disease of the nervous system as it occurs in the whites, nevertheless, the clinical syndrome in each case precludes the serious consideration of any other disease. The cases tabulated here were seen in Dr. H. T. Patrick's clinic, Northwestern University Medical School.

CASE I. (Referred from Eye Department, Northwestern University Medical School, by the courtesy of Dr. Richard S. Patillo, who anticipated the possible diagnosis of tabes fom the "fundus" findings.)—February 4, 1903, M. R., aged thirty-nine years, female, colored, married. Born in Louisville, Ky., of black father (slave) and bronze mother (slave). The father lived to grow very old at the Soldiers' Home, Wyandotte, Kansas; cause of his death unknown. Mother lived to the age of sixty-five years and died of some asthmatic trouble.

The patient, as a young girl of twelve years and upward, was subjected to exposures of wet and cold, which led to the development of rheumatic symptoms that have prevailed from year to year with moderate severity. The hardest of work was required of her in an attempt to support the family. One brother died of smallpox at the age of twenty-three years, and one sister died of pneumonia at eighteen years. At the age of sixteen years the patient miscarried an illegitimate child at four months. She attributes this occurrence to a fall which she experienced at the time. At eighteen years she married the father of her first illegitimate, and gave birth to a full-term, living male child in the first year of her marriage. Prior to the birth of the child she records having had a protracted siege of sore throat, and adds that the husband abandoned her, with the excuse that he was afraid of giving her some "bad disease." She denies knowledge of specific taint, and upon inquiry records no signs of secondaries. I detail the data of ancestry and venereal infection, holding them to be particularly important in the anamnesis of these cases. At the age of eighteen years she had what she pleases to call an attack of pneumonia, but since that time and up to the date of onset of the present trouble she has had "no misery nor corruption anywhere."

Present Illness. Onset about five years ago, with severe headaches persisting for a week at a time, disappearing and recurring for longer or shorter intervals. Headaches agonizing, occipital, vesperal. Simultaneously, dimness of vision in left eye, which glasses did not seem to relieve. Failing vision progressive until two months ago, when she noticed complete blindness in the left eye. Sight in the right eye began to fail shortly before the vision in the left eye was destroyed, and is becoming more and more impaired. Early in the course of the trouble patient experienced severe paroxysmal pains in the stomach, coming on once or twice a week, more often at night, localizing with greatest intensity at the navel. Describes sensation of a rope tied with increasing tightness around her middle. Refers to leg pains as boring and gnawing in character, beginning at the ankles, ascending to the knee and fleshy part of thigh. Sense of tingling and of "things crawling" on the soles of the feet. Of incoordination it may be said that the patient is occasionally inclined to stumble or stub her toe against the floor, especially at times when she feels nervous. Frequently finds it necessary to hurry to the toilet when she least expects it, for fear of soiling her clothes. Despite the good appetite which she enjoys, she has lost about twenty-five pounds in weight during the past year. (This fact, together with that of a temperature of 100.5° F., led me to suspect a tuberculous focus, which the physical examination revealed as present in the left apex of the lung.) Bowels constipated; sleeps fairly well; says she is more concerned about her eyes than all the other symptoms.

Status Præsens. In general appearance of average height and fair nutrition; bronze-skinned; mouth and pharynx negative for post-specific scars; heart negative; no peripheral arteriosclerosis; pulse regular and full; lungs give evidence of left apical tuberculosis; urine negative; glandular system, isolated enlarged glands, especially inguinal. No bony deformities. Sexual instinct not so keen for past five years—has had " little nature for a man."

Sensations. Subjective: Tingling and formication on plantar surfaces of feet. *Objective*: (1) Tactile sensibility everywhere acute; (2) well-defined analgesia, both legs from knees down; (3) trunk anæsthesia absent; (4) ulnar analgesia slight. No trunk anæsthesia. No persistence of painful impressions. No delayed conduction of sensory impressions.

Sphincters. Slight *incontinentia urinæ.*

Coordination. Virtually unimpaired; no ataxia of gait; no ataxia of station except when put to the test of balancing on one foot. Finer tests, such as heel to knee and finger movements, show slight degree. Tremors absent.

Reflexes. Patellars bilaterally absent; Achilles, bilaterally absent; plantars exaggerated; abdominal, upper and middle zones brisk; Babinski absent; wrist absent.

The Eyes. I am indebted to Dr. Richard S. Patillo for the report of the eye finding, as follows: Total iridoplegia; no oculoplegia. Visual field with " white," one-half inch square; above, two inches; nasal side, two inches; temporal, one inch; lower, three inches; apparently no scotoma. Ocular tension normal. Double optic atrophy. Acuity of vision, O. D. $\frac{20}{200}$; O. S. $\frac{00}{00}$.

CASE II.—L. M., aged forty-six years, female, colored, married; color light bronze. The ancestry of this patient is as follows:

Paternal grandfather, full-blooded Indian; paternal grandmother, full-blooded negress; maternal grandfather, full-blooded negro; maternal grandmother, Irish. Thus these strains make this woman one-half negro, one-quarter Indian, one-quarter Irish. Patient born in Ricetown, Maryland, and reared in Baltimore. Parents were free when patient was born.

Of past illnesses, patient records croup and measles when a child, and typhoid fever at sixteen years. Married first at age of nineteen years to a full-blooded negro. Four years married, when she miscarried at two months—no ascertainable cause. First husband died. Married again at age of thirty years, also to a full-blooded negro; second marriage childless. Has no knowledge of a luetic infection. She does, however, state that after the first year of married life, that is, at the age of twenty years, she experienced severe " sick " headaches, coming on with increasing frequency at any time of the day, locating with greatest intensity over the right eye, and again over the left, and accompanied by a sense of nausea, which would occasionally end in vomiting.

Present Illness. Began in 1895, with severe throbbing pains in the legs, neuralgic, as she calls them, and along the nerves, radiating from the thigh downward into the calves. Vision began to fail in 1896. She at that time was under treatment at the Illinois Eye and Ear Infirmary, and was referred by the oculist to Dr. Patrick, who saw her for the first time at the Polyclinic in that same year, and made a diagnosis of tabes. Sight in the left eye was completely lost within six months from the date of onset. At this time incoordination was not very marked. The right eye began to fail her soon after the left, but for the past four years vision has remained stationary. She claims that she can see the daylight out of the corner of the left eye (mental); with the right eye she can distinguish daylight from lamplight or differentiate a bright from a dark day, but she cannot see to read—in other words, she has only "quantitative vision."

Status Præsens. In appearance is fairly well nourished; of bronze complexion; mouth and pharynx normal; thoracic viscera normal; urine negative; sexual instinct indifferent.

Sensations. Subjective: Early in the disease as of "cold water running under the skin," but of late this has disappeared. *Objective:* (1) Tactile sensibility everywhere most acute; (2) analgesia in the limbs, slight from the thigh to the knee, but very marked from the knees downward; (3) trunk anæsthesia absent; (4) ulnar analgesia present.

Reflexes. Patellar: Left abolished, right very faint response with reinforcement; plantar reflexes normal; Babinski absent; wrist taps present; abdominal and skin reflexes present to slight degree.

The Eyes. I am obligated to Dr. Salinger for the eye-findings in this case. Quantitative vision only. Total iridoplegia, both disks gray-white and excavated; double optic atrophy. No ocular paralysis.

Coordination. Gait unimpaired; no static ataxia; slight ataxia to finger movements. Sphincters: Slight *retentio urinæ.*

CASE III.—W. F. P., male, black-bronze in complexion, aged forty-nine years, married these ten years—childless marriage. By occupation a waiter. I was unable to ascertain so exact an heredity as in the previous cases. I did determine, however, that the father died an old man from some kidney disease. The mother is living and well, as are two sisters and one brother.

The patient had measles as a child. States that he had gonorrhœa as many as five or six times, the last attack eight years ago. No history of chancre. He has been temperate in his habits, drinking beer moderately, and does not deny taking an occasional whiskey, as he says, to stimulate him since his present nervous trouble has come on. As to injuries, he presents a scar over the left temporoparietal region, which he states is the result of a lacerated wound of the scalp received some twelve years ago from a glass of water thrown at his head.

Present Illness. Onset about four years ago, with dizziness, especially distressing when the patient got out of bed in the morning, and which in a short time reached such an alarming degree as to make his efforts at walking for the first two or three hours of the day almost impossible. In his own words, he could never "gather himself together until about noontime." (I believe that this dizziness is to be regarded more in the light of incoordination than vertigo.) He says that frequently his swaying from side to side was so great as to give onlookers the impression that he had been drinking. During the latter half of the day he grew steadier on his feet and kept so until the following morning, when the evidences of his incoordination promptly reappeared. A loss of memory has been a conspicuous feature in this patient's case from the very first. Being a waiter by occupation, he found himself greatly handicapped by his poor memory in receiving and remembering orders given him. He would forget parts of an order, and lately he has taken an order as far as the kitchen only to return and ask the customer to repeat same, as he had entirely forgotten it. About one year after the onset of this dizziness and impairment of memory his eyesight began to fail him—three years ago. With this failure of vision there has come a sense of fear when the patient is abroad (born of his ataxia), having fallen time and again over such objects as chairs and other obstacles that seemed to have been in his way. As he puts it, he has "lost his nerve." For the past seven months he cannot see well enough to read print. Fulgurant pains radiating from the thighs into the calves have been present for the past two and one-half years. The patient has regarded these as rheumatic. In addition, he complains of a sense of tightness at the waist, which impression he, of his own accord, chooses to disabuse me of by saying that his trousers are far too large at the waistband to account for this feeling. There can be no doubt that this sense of constriction is none other than a "girdle sensation." Sexual power is weakened to a degree of partial impotence. Has lost appreciably in weight, despite his good appetite. Bowels constipated.

Status Præsens. General appearance that of a man in good nutrition; weight, 145 pounds; height, 5 feet 1 inch. Heart and lungs are normal; no vasomotor disturbance; no peripheral vascular changes.

Sensations. Subjective: Soreness and a sense of burning on the soles of the feet. Says that at times he feels as though he were walking on a gravel path. *Objective* sensations, negative.

Coordination impaired, but only slightly; most manifest when told to turn quickly or suddenly rise from his chair. Not at all present during his more deliberate movements. Romberg sign absent. Slight tremor (coarse) in the right hand upon protrusion.

Reflexes. Left patellar brisk; the right almost abolished. Contrastingly, the left Achilles is abolished and the right Achilles is faintly present with reinforcement. The abdominal skin reflexes are everywhere brisk.

Eyes. (Examination by Dr. R. S. Patillo.) Pupils equal in size, the left slightly irregular in outline. Right pupil reacts to light sluggishly; left pupil does not, but both react to accommodation. Fundus: Optic atrophy in both eyes. Acuity of vision: O. D. $\frac{8}{120}$; O. S. $\frac{00}{00}$; ocular tension normal, no oculoplegia.

Organs of special sense uninvolved. Tongue shows neither deviation nor tremor.

Referring once more to this patient's complaint of dizziness, I do not wish to insist that the patient could not have been suffering from a genuine vertigo in the incipient stage of the disease, because it may be and sometimes is a feature in some cases, but is of distinctly inferior importance and occupies a low place in the scale of conspicuous symptoms. Bonar[11] in 286 cases found it in 0.34 per cent. Another rare symptom presenting in this case is that of tremor, which Bonar[11] found in 1.74 per cent. of his 286 cases.

CASE IV. (in brief).—S. M., female, aged forty years, light brown in complexion (lightest in complexion of all the cases), born of black mother and white father; married and has one living child, prior to the birth of which the patient had two miscarriages, without ascertainable cause. Seamstress by trade. Knows nothing of father; mother living, but patient does not know where. Has one brother and two sisters, all of whom are even lighter in complexion than she is.

Patient had typhoid fever at the age of eighteen years; also vaguely refers to fever and chills. Onset of present illness six years ago, with severe headaches and excessive nervousness. Inquiry as to this nervousness elicited the fact that she had difficulty in threading her needle, although she could see the eye of the needle as plainly as ever before. There were days when she was not annoyed by this symptom, and then for weeks at a time the symptom would reappear. When using the machine her line of stitching was anything but straight at times, and her limbs easily gave out in the act of treading. The symptoms continued until she was forced to give up this kind of work. In the third year of her trouble, eye symptoms developed and for a short period of time prior to the beginning failure she saw things double (diplopia). Bladder disturbance manifested itself about this time. She gives no evidence of disturbance of gait or of severe lancinating pains. She is more worried about her eyesight than anything else, and deplores her inability to resume work as a seamstress. Her symptoms have not taken on an increasingly aggravated form for the past three years. Denies venereal infection.

Status Præsens. A female of slight build, about 110 pounds in weight, and taller than the average woman. Yellow skin; somewhat emaciated in appearance; heart and lungs normal. Urine negative.

Sensations. Subjective: Cramp-like pains in the calves, paroxysmal, but not severe. *Objective:* Analgesia most marked on the dorsal aspect of the feet.

Coordination disturbed and easily detected upon an attempt at finer movements; cannot approximate index fingers well, nor can she grasp small objects without very apparent awkwardness. No ataxia of station except when standing on either foot alone. Gait normal. Hands, more especially the fingers, tremulous upon protrusion.

Reflexes. Both patellars completely absent. Achilles both absent. Abdominal reflexes and skin reflexes faint in their response. Babinski reflex absent.

The Eyes. Pupils equal in size and regular in outline. The irides do not react except to accommodation. Fundus: Partial optic atrophy in both eyes. No ocular paralysis.

Complains of deafness in left ear, which, however, she says is not constant.

I hold it would be erroneous to attach undue importance to the conclusions that accrue from an analysis of so few cases, but that fact should not deter from bringing out and emphasizing points of difference and similarity in the disease as it affects another race than the Caucasian. In the matter of sex the most recent and conservative figures imply a proclivity for this disease in the Caucasian male, and it is a matter of no little interest that in the colored race, respecting my four isolated cases, the frequency is quite the other way. As to the age at which the disease manifests itself in these cases, it concurs with the statistics that have placed more than 50 per cent. as occurring in the third decade.

The ocular phenomena in this series merit special attention. One of the cardinal symptoms of tabes lies in the condition of the pupils. The Argyll-Robertson pupil was present bilaterally in three of the cases, and in the fourth was found in the left eye, there having been a sluggish reaction to light in the right. The intense pigment in the eye of a colored individual renders a decision as to the presence of a pupillary reaction and its degree of prominence much more difficult than in the eye of a white person. The pupils in all but one case were equal in size and regular in outline. Schaffer,[14] is authority for a remark made by Gowers that the Argyll-Robertson pupil is only present in those cases of tabes which are of undoubted luetic origin. I am not aware that other clinicians coincide with this statement. The failure of vision occurring in all the cases, revealing for its cause the presence of optic atrophy in all of the eyes, led me to look into the question, and I found that relative to the statistics of optic atrophy in tabes as it afflicts the whites there is great variation. Von Grosz[15] found it present in 88 per cent. of his cases; Berger,[16] in 46 per cent.; Dillman,[17] in 42 per cent.; Marina,[18] in 12 per cent.; Bernhardt,[19] in 10.3 per cent.; Thomas, 10 per cent.; Leimbach, in 6.75 per cent. Bonar's figures of 8.74 per cent. are based upon an exhaustive and critical analysis of 286 cases of tabes from the case records of Allen M. Starr's service at the Vanderbilt Clinic, New York. Bonar states that optic atrophy was either well established or beginning in 25 cases, "and," he adds, "one case was that of a colored man in whom the atrophy came on as the first and only symptom except the blindness caused by it and the absence of the knee-jerks." The well-known fact that cases of profound tabes so often come under the care of the oculist for disturbances of vision, and are by him referred for a wider interpretation to the neurologist, led me to investigate ophthalmological statistics for data as to the comparative prevalence of optic atrophy among the whites and blacks.

In the *Archives of Ophthalmology*, 1884, Swan Burnett's[21] treatise on the "Comparative Frequency of Eye Disease in the White and Colored Races of the United States" included a consideration of the diseases of the optic nerve, retina, and glaucoma. His statistics comprehend a consideration of 2325 cases, of which 1514 were colored and 811 were whites, and all of which were by him personally examined and observed. He subdivides his cases into diseases of the conjunctiva, diseases of the cornea, diseases of the iris and choroid, etc., diseases of the optic nerve, retina, etc., as follows:

DISEASES OF THE OPTIC NERVE, RETINA; GLAUCOMA. (SWAN BURNETT.)

	White.	*Colored.*
Atrophy of optic nerve	14	13
Retinitis albuminurica	2	5
Amaurosis	1	7
Retinitis sympathetica	...	1
“ specifica	3	3
“ pigmentosa	1	2
Neuroretinitis	...	3
Myxoma, optic nerve	1	...
Glaucoma, simple	4	14
“ inflammatory	1	2
“ secondary	...	1
Hyperæmia retina	2	2
Glioma retina	...	1
Detachment	2	...
	31	54

He fails to comment upon the finding of optic atrophy as a symptom of tabes—in fact, he does not discuss the matter at all. It would be interesting to know what other oculists have to report in this connection.

The cases reported by McConnell[22] in 1899, instances of tabes in the full-blooded negro (according to the author), were of the amaurotic type. In his cases, as well as in mine, all the symptoms, some of which had attained an aggravated degree, were held in abeyance as soon as the optic atrophy had manifested itself. Benedickt[23] goes so far as to say that he had never known an exception to this condition, and Bonar,[24] relative to his twenty-five cases, in which the atrophy dominated the other symptoms, says: “Where the first symptom was the blindness due to optic atrophy there has been no further advance of the disease.”

Abeyance of all other symptoms naturally includes an arrest of incoordination, which actually occurs. It is a rare thing for a man with tabes to have marked incoordination and almost total blindness —one or the other may be conspicuous, but seldom, if ever, coexistent. As Dr. H. T. Patrick,[25] in a clinical lecture on “Locomotor Ataxia,” says: “This is not a trivial matter, because one patient consulting us for trouble with his legs may know another patient with locomotor ataxia who has become blind, and *vice versa*, and he is greatly worried for fear he may acquire the same symptom. If a patient comes to you with failure of vision from locomotor ataxia, you are justified in assuring that patient that he will always have good use of his legs.” Just why this is so has been for years a matter of conjecture. When McConnell presented his five cases before the Neurological Society of Philadelphia, in 1899, emphasizing the abeyance of ataxia in the presence of the optic atrophy, it was suggested in the discussion to invoke the Edinger compensation theory (Ersatz theorie)[26] in explanation of this conciliatory effort on the part of the damaged nervous system. Dr. Spiller, in the discussion, stated that he had entered into an interesting correspondence with Edinger on this point, and could quote a passage from his (Edinger's) letter, which read to the effect that the substitution theory did not supply *all* the needs in the cases to which it applied.

For those less familiar with the Edinger hypothesis, published in 1894, I have given a succinct interpretation of the principles therein involved.*

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* Edinger's theory is based upon the time-honored physiological dictum which was original with Weigert, and states that the usage and function of a part implies disintegration and consumption of some of the active tissue. In this sense it has been applied to the nervous system by Edinger, who furthermore assumes that where nutrition in a part is deficient (or even under normal conditions) and the cells are called upon to do excessive work, suprafunction, thus disturbing the balance between combustion and repair, the developmental energy in the resting tissue will serve to bring about a degeneration of the less resistant active parts.

In order that the Weigert-Edinger view as it pertains to both motor and sensory spheres of the nervous system succeed it is essential that it be proved that certain tracts under normal conditions are called upon to perform more work than others, and that these overworked tracts undergo correspondingly pronounced degrees of degeneration. Edinger regards as a prerequisite to the decline a disturbance of general metabolism and a deficient nutrition.

It is observed by Edinger that the sensory nerves, for instance, are constantly transmitting sensations from the skin to the central nervous system; the fact that we are unconscious of the presence of a sensory mechanism does not mean that we are not possessed of one in constant operation, for we are, and the sensory nerves are the very ones, for that reason, to suffer.

From these hypothetical premises are argued the production of the clinical sensory symptoms—anæsthesias, paræsthesias, hyperæsthesias, etc. The sensory disturbances evidence themselves, as might be expected, in those areas on the body where the peripheral nerves are most irritated from contact, for instance, with or pressure of the wearing apparel. Corroborative of this reasoning, there are found in tabes irregular and scattered patches of anæsthesia occurring frequently on the soles of the feet and in a narrow band encircling the waist. This ingenious reasoning Edinger applies to the eyes, more especially to the nerves of the "constrictor iris," since there can be no glance at any object, no matter how momentary, without this muscle and its nerve supply coming into play. The light reflex is almost constantly active, in contrast to which the accomodative reflex is but seldom so.

Reverting once more to the physiological principle, we might expect a degeneration of the fibres controlling the reflex arc to light, while the latter, those governing accommodation, remain normal. In this way the author of the theory chooses to explain the clinical phenomena known as the Argyll-Robertson pupil, optic atrophy, oculoplegias, and especially ptosis. The disturbance in function of bladder and rectum are alluded to in the same sense.

Edinger has drawn a parallel between the above symptoms ascribed to suprafunction in an ill-nourished nervous economy and those which complete the typical picture of tabes, viz., the incoordination, lost knee reflexes, Argyll-Robertson pupil, oculoplegias, optic atrophy, and loss of sphincter control.

If his deductions are correct, the theory supplies a more satisfactory explanation of the pathology of tabes than any as yet offered, and better accounts for the presence of many hitherto less well understood but widely recognized clinical phenomena.

Profound study of the hypothesis thus far constructed led Edinger to carry on experiments in furtherance of the truth of his ideas. He followed in the footsteps of Voss, who produced extreme malnutrition in animals by the use of substances (pyrodin) which impaired the blood-making powers, and then subjected them to forced activities. Animals so treated showed upon post-mortem examination degenerations closely resembling those of tabes in the motor tracts of the cord.

By inference, then, it is clear that Edinger's theory postulates (1) the deleterious effect of noxious agents on the nutritional state of the nervous system; (2) a nervous soil so prepared is further agitated by normal or excessive function until a symptom-complex results, such as we are, for instance, familiar with in tabes.

The theory met with an objection at the hands of Schultze, who argued that if function determined the localization of the disease there would be every reason to note degeneration in the motor nerves and anterior cells, with occurrence of paralysis and muscular atrophy. This objection was overruled by Edinger with the assertion that the motor nerves were ana-

That the Edinger theory as it applies to both motor and sensory spheres of the nervous system succeed it is essential that it be proved that certain tracts under normal conditions are called upon to perform more work than others, and that under the influence of undue stress in the form of overwork, either relative or absolute, these tracts degenerate, this degeneration being associated with a disturbance of general metabolism plus deficient nutrition. In other words, given a man who is drenched with substances of noxious character, toxins, if you will; acknowledge that his entire economy has undergone a metabolic change as a result of their influence, and let him use to excess such faculties or functions as involve certain tracts at the expense of others, and *there* the deficit is most felt, and *there* the degeneration is most evident. People who use their lower extremities most are prone to suffer from ataxia in the legs; people using their eyes most have to contend with optic atrophy. In the course of human events it would be ridiculous to aver that the negro uses his eyes at the expense of his legs or other organs; in fact, it is perfectly right to assume that he uses them far less. Here we encounter a weak point in the Edinger hypothesis. And Edinger himself, true and fair-minded scientist that he is, acknowledges the fallibility in part of his reasoning when he states that his theory may not cover all the needs of a case. There is an experimental groundwork for the substantiation of Edinger's theory, even though the clinical data are not always corroborative. These experiments are highly

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tomically safeguarded with a greater power of resistance and a better supply of nourishment than the sensory.

With an acceptance of the Edinger theory much that is vague and indefinite as to the tabes-syphilis question becomes satisfactorily explainable. The normal relation between waste and repair is influenced by the noxious action of syphilis on all the tissues of the body and further impaired by the coincident vascular changes of the disease.

Acknowledging the high percentage of tabetics who are known syphilitics, one is enabled to account for those cases which give neither the history of specific infection nor somatic evidence thereof. By this token we are not called upon to believe that everyone who has tabes must have had syphilis—a fact insisted upon by some authors, but still unproved by reliable statistics.

Edinger has provided a unitary etiological factor for the disease tabes in that syphilis, exposure to cold, wetting, trauma, excesses in "Baccho et venere" are grouped as predisposing noxious features disturbing the nutrition of the nervous apparatus, the associated undue stress of function completing the requirements of the clinical picture.

Even the clinical symptoms bear out the theory: the lower extremities are affected first in a majority of the cases, and in the few cases of tabes high up in the cord, the cervical type or the *tabes cerebrale* of the French, the information was elicited by Edinger that the arms had been called upon to do proportionally more work, and that these patients were by occupation seamstresses, carpenters.

The sex ratio as variously established, from 15 or 3 : 1 in favor of the men, fits well with the theory, since men engage in physical pursuits.

In referring to the pathological process and its limitation to the intramedullary distribution of the sensory neuron and not its peripheral part, which is virtually exposed to the same stress, Edinger explains that (1) the peripheral nerves enjoy the protection of an additional sheath; (2) the least resistant point of the neuron, owing to a constriction of the myelin sheath (or its absence), is at its point of entrance into the cord.

As for a consideration of the therapeutics applied in consonance with this theory, suggestions have been made in the body of this paper.

instructive. By feeding rats pyrodin he caused a profound intoxication and anæmia, after which they were made to indulge in great activities of a certain kind, and it was found that those rats that had been put hard at work showed, post-mortem, a correspondingly advanced grade of spinal-cord degeneration. This discovery led Edinger into the field of therapeutics with a suggestion in prophylaxis, "insisting that exertion and fatigue of any sort should be avoided," maintaining further that, "in a large number of cases treated for a number of years on this basis, results have been obtained in the way of retarding the progressive development of the disease." The proportion of males over females he attributes to this same pernicious effect of overactivity.

I deem it expedient at this point to introduce evidence of very recent date and from a most reliable source, which, to my way of thinking, harmonizes excellently well with Edinger's experimental findings and therapeutic results. A no less distinguished clinician and observer than Marie is responsible for the statement that in his amaurotic cases, in which, *intra vitam*, the ataxia and other symptoms were arrested, post-mortem examination revealed a *less extensive* degeneration in the cord.* Edinger's claim is that bodily overaction increases the degenerative process in the cord and that bodily inaction restricts it. Marie by another route, namely, that of blindness, determines substantially the same fact. In the presence of these data, then, how much nearer are we to a solution of the question, Why does established optic atrophy retard ataxic symptoms? Time was, and only a few years ago, when J. K. Mitchell[27] offered the following explanation: "The blindness does result in a certain concealment of some of the tabetic symptoms—the blind patient, if he can walk at all, necessarily does so without seeing his feet; thus, perforce, he acquires some of the greater ease of motion and improvement in station which the tabetics acquire by learning to execute careful coordinate movements—*not that they have changed the disease tissues of the cord, but* that they use themselves enough better to conceal some of the symptoms of the disorder." Since then Marie's observation has reinforced Edinger's view, and out of the combined issue I believe it is possible to advance a more tenable assertion. The patients with optic atrophy are blind; blind men rather conserve than dissipate muscular energy. They do not overexercise. With them functional wear and tear is at a minimum, and so the disease process in the cord is restricted. A less degenerated cord reciprocally inhibits the other "motor" symptoms. As to why the optic nerve is selected to atrophy in the first place, see note appended on the Edinger compensation theory.

The medical profession has had the Erb-Fournier parasyphilitic postulate so thoroughly impressed upon it that it is unwilling to credit any other factor as giving rise to tabes. Some regard, but very little, is shown for von Leyden's conception invoking exposure to wet and cold, and *not* syphilis, as the chief factor in the cause of the disease. Only the fewest are disposed to regard Edinger's theory favorably, although it explains more of the multiform symptoms than any other one hypothesis. Benedickt, who, when he says "*tabicus non fit, sed nascitur*," emphatically denies every factor but that of heredity, meets with little approval at the hands of the profession.

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* A personal communication of Marie to Dr. H. T. Patrick.

Summing it all up, this much is incontestable, that tabes is a disease of the sensory neuron, caused, in a vast majority of the cases, by syphilis, which, however, does not stand in direct causal relation.

Coming to a consideration of the cardinal cause in the production of the disease in the negro, and conceding an apparently relative immunity in him, who shall deny that Aryan admixture is not accountable for a predisposition in the man, which, plus his vices and acquired venereal infections, has given rise to the appearance of tabes in him? In support of this view I recall that all of the cases heretofore reported, including my own, in which I have been especially solicitous of detailing the ancestry, occurred in hybrids. McConnell, it is true, reports his cases as those of full-blooded negroes, but adopts the view from the premises of color rather than by inquiry into ancestry. A black negro need not necessarily be a full-blooded negro, and I am not willing to allow that mere observation with respect to varying shades of complexion is convincing.

Conspicuous as is the part played by venereal diseases in the life of the negro, syphilis, so very much more prevalent among the colored, is on the increase, and facts supplied from various authentic sources go to prove that the negro has acquired this infection since the period of slavery through an ever-increasing low standard of sexual morality.

While it has proven futile to insist upon a syphilitic etiology for tabes in the Caucasian, nevertheless, I think a causal relationship as it exists for the whites applies in the blacks.

The query as to why there is so little tabes in the negro when there is so much syphilis is a natural and logical one, which may remain unanswered for the same reason that other paradoxes of factual medicine exist and solution of them is suspended. As well might we seek to explain the absence of tabes in Bosnia and Herzegovina, where, as we know, the most intense constitutional syphilis is rampant among the natives. So far as immunity and susceptibility is concerned, I believe that too much has been taken for granted.

As Dr. Matas[8] concludes, "there are no diseases which prevail exclusively in the colored any more than there are diseases which prevail exclusively in the white race."

If any conclusions are to be drawn from a consideration of the various features touched upon in this paper, they may be summarized as follows:

1. Long residence with the white man has made the American negro anthropologically, physiologically, and pathologically different from his African ancestors.
2. The constitutional variation has been wrought by acclimatization, social environment, and, more than all else, by miscegenation.
3. The influence of miscegenation and the advent of personal liberty are responsible for a new era of diseases.
4. The newer diseases in the negro, of which tabes is an example, are fast becoming more commonly recognized, miscegenation being regarded as the potent factor in reducing the negro's resistance toward disease.
5. Tabes exists in the negro perhaps more commonly than has been supposed, and failure to recognize it may be due to the abeyance or total absence of the ataxic symptoms in the amaurotic type.
6. The Edinger-Marie observations anent the optic atrophy satisfactorily explain that class of cases in which the tabes is arrested by blindnese.
7. Aryan admixture is essential to the production of tabes in the negro.

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